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Synopses

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The effects of medication on oral health. Myths and realities

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"First do no harm"
Hippocrates, 460-355 BC.

Introduction

The use of medications (a substance taken internally to preserve or restore health) is a common occurrence for many children. Myths (popular beliefs or ideas) such as children's teeth being soft, weak and discoloured due to medications, milk builds stronger teeth or beliefs such as taking certain medications during pregnancy strengthens teeth, or that bad teeth are due to unfluoridated water and aspirin next to a tooth relieves a toothache are some examples that will be addressed.

As medical science progresses, medically compromised children survive longer and more children are on medications. Clinicians must be familiar with their effects on oral health so that sound clinical judgement can be used to advise or possibly alleviate any resulting oral pathology. Realities regarding the injudicious use of medications, medication-induced gingival overgrowth, effects of medications used in oncology or transplants will be addressed.

Myths

MYTH: "My sick child's teeth have become soft and slowly eaten away or are weak and easily chipped due to the long term medications he is on".

There are various reasons for these parental lay descriptions of the child's teeth. Medications can contribute to dental caries, xerostomia, changes in saliva[28] and enamel hypoplasia and opacities.[40]

Dental caries

The multifactorial aetiology of caries is based on the relationship between host, bacteria, substrate and time.[26] Fermentable carbohydrates (substrates) such as sucrose, glucose or fructose are added as sweeteners to many chewable or liquid pediatric pharmaceutical preparations to enhance palatability and compliance.[12] These sugars are used as diluents, preservatives, wetting agents, demulcents and viscosity-modifying agents.[26] Syrups can contain up to 8g of sucrose per 10ml, or have a 30% to 70% sugar content.[11,33]

Evidence in the literature implicates fermentable sweeteners in liquid or chewable medications in causing caries.[11,12,26,27,38] A study by Feigal et al tested the cariogenicity of seven sweetened liquid medications and found that many had a high sucrose content and also that the intraoral pH response equaled or exceeded that seen when sucrose rinses were given alone[12]. They concluded that the high concentrations of fermentable carbohydrate in medications could lead to high acid production.[12]

Observational studies have shown a high caries prevalence in children on long-term liquid medications.[11,27,38] Roberts et al found no other factors related to diet or dental health practices that could account for the increase in caries incidence.[38] However, Feigal et al found that only a small number of young cardiac patients on sweetened liquid medication had very high caries experience, suggesting that it does not universally lead to high caries rates.[11] If the study was done on a larger population, the effect on caries rate may be more significant.

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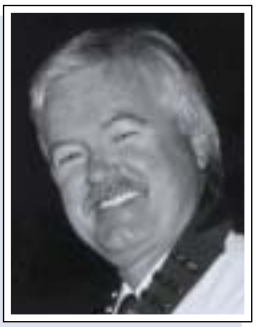

President's Report

AN EXCITING AND PRODUCTIVE YEAR

I hope all members had a happy and healthy Christmas and New Year break. I for one have found it difficult to regain my pre-holiday work routine and commitment. A recent visit to Sydney to assist with the examination of candidates for fellowship of the Royal Australasian College of Dental Surgeons helped to dispel any ideas of continued leisure activity.

The level of scientific knowledge and clinical skill that the participating dentists demonstrated in the section of paediatric dentistry and orthodontics was exceptionally high. The college has always maintained a high standard in this particular field and ANZSPD can be justifiably proud of those members that elect to participate in this grueling exercise.

My congratulations to Drs Helen Cornwell, Soni Stephen, Nina Vasan and Jemima Roberts for their recent admission to general fellowship within the college and special mention to Nina for winning the Kenneth J G Sutherland prize for the highest mark in the general dentistry section of the examination.

Whilst on the subject of examinations, the number of students in paediatric dentistry within Australia and New Zealand completing their postgraduate training has shown a steady and encouraging increase in recent years. I counted seven graduates last year and hopefully they are now preparing to enter the dental workforce this year. My congratulations to all successful candidates. Almost everyone acknowledges that we need more paediatric dentists to help meet the special needs of children. However, even with a modest increase in paediatric dental training positions, improved workforce distribution and better management of public oral health programs, we will not be able to solve the national problem of early childhood caries alone. We also need help from non-paediatric dentists to help manage those children who are prepared to treat these children in general private or public practice.

It amazes me that another society year has passed so quickly. Even more amazing is the amount of time and effort that the secretary manager has devoted to our business during this time. The results of his labour are clearly shown in the increasing volume of inward and outward correspondence and general housekeeping that Alistair has to deal with on our behalf. Alistair is currently exploring the option of incorporation of the society to facilitate the organisational framework required for the IAPD Congress in Sydney, 2005.

Early Childhood Caries Symposium sponsored by Colgate Oral Care on Monday 7 May

The federal executive has agreed to establish a separate legal entity to support the IAPD meeting, thereby insulating the current structure from any financial shortfall. It will be necessary to establish a separate bank account and engage a conference organiser for this purpose. IAPD President, Dr Richard Widmer and his organising committee have already commenced the planning process in earnest. Members who can provide any help and administrative support would be greatly appreciated, even at this early stage. Please contact Richard directly with your ideas.

Closer to home, the 30th Congress of the Australian Dental Association will be held in Brisbane from 4-8 May this year. I hope that all ANZSPD members will be able to attend the congress and more particularly, the Colgate Oral Care sponsored Early Childhood Caries symposium on Monday 7 May. ANZSPD has selected range of speakers to present current scientific and clinical information in their relevant subtopics. Brisbane is at its best in May with mild sunny days and temperate nights for those members that wish to take advantage of the outdoor social and leisure program.

Finally, my sincere thanks to the Western Australian branch members for hosting my visit to Perth last year. The pace was somewhat frantic, but most enjoyable, and it was surprising how much activity can be squeezed into one long weekend. I hope to be able to visit each branch at least once during my presidential term and look forward to meeting with all members in due course.

Regards and best wishes for the new year.

Kerrod B Hallett

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Maguire et al found that children on long-term liquid medications had more caries on their deciduous anterior teeth than their siblings.[27]

The impact of long-term antimicrobials on caries remains unclear[27]; they may actually be cariostatic[20,23]. A study by Kinirons to determine the effect of antibiotic therapy on the oral health of children with cystic fibrosis found that those on medium to long term antibiotic therapy (not stated if the medication had sweeteners) had lower prevalence of caries, plaque deposits and gingivitis than age-matched controls.[23] Karjalainen et al studied the long-term effects of antimicrobial syrups for recurrent otitis media on the dental health of 6 to 8 year old children, and found a decline in the prevalance of caries when compared to age-matched controls.[20] The ability of long term antibiotics to reduce the cariogenic challenge is explained by the effect on cariogenic bacteria, especially mutans Streptococcus.[20,23]

Asthmatics using powdered inhaler devices or aerosols have more caries, poorer periodontal status and more tooth surface loss then healthy controls.[15,19,28,30] These devices use a lactose carrier to assist taste recognition of the medication (up to 25mg of lactose per dose). Although lactose is less cariogenic than sucrose, its use when coupled with the reduced salivary flow of asthmatic children (a side effect of beta 2 agonists) contributes to caries. These children may also be taking sweetened medications for their upper respiratory tract infections. They may be more thirsty than their counterparts due to reduced salivary flow, and parents may also be more indulgent in giving cariogenic drinks and sweets.[19,28]

The poorer periodontal status in these children may be explained by their altered immune response (due to inhaled steroids) and their tendency to mouthbreathe, especially during asthmatic episodes and rhinitis[28]. These children also have more erosion due to a variety of causes, including the inhaled powdered drugs (pH less than 5.5), so patients should be instructed to rinse their mouths with water following drug administration[30]. McDerra et al have stated that since erosion is multifactorial in nature, further research is needed to investigate this implication[28]. In a study to determine the effects on saliva and plaque pH in asthmatic children, decreased salivary and plaque pH was evident after the use of beta 2 agonists[19]. Therefore, asthmatic children may be at higher risk of developing caries due to their medication.

The Canadian Dental Association (1999) has proposed prescription guidelines because of concern over the use of sweeteners in medication[2]. However, no guidelines exist for Australia or New Zealand.

There is evidence to prove that medications with fermentable sweeteners cause caries. It is important to note the mode (chewable, inhaled or liquid), frequency and time (such as just before bedtime when salivary flow is low) at which they are administered, their high viscosity, and the type of patients receiving these medications (infants and special needs patients with poor oral hygiene habits or those without regular compliance with toothbrushing especially after drug administration) may contribute to their high caries rates.[2,11,33]

Xerostomia

Saliva (via its lubricating, cleansing, remineralising, antibacterial and buffering actions) is a protective factor for both hard and soft tissues[3,31]. The main causes for xerostomia (reduced salivary flow) are drugs, irradiation and diseases such as Sjogren's syndrome[3,18]. Medications with anticholinergic activity such as sedatives, antipsychotics, anti-depressants, antihistamines, antireflux medications and diuretics are associated with salivary gland hypofunction[3,14]. In fact over 400-500 medications are associated with xerostomia.[3,14]

The effects of xerostomia on the oral cavity include dental caries, especially along the cervical margins and incisal edges of teeth, and an overgrowth of Candida species. Salivary glands may be affected by recurrent, retrograde bacterial infections; patients have dry lips, mucosa (with mucositis) and tongue, and they may be predisposed to oral lichen planus and periodontal disease[3,18,31]. They may complain of disturbed oral sensation and taste, speech dysfunction, halitosis and swallowing difficulty[31,39]. Xerostomic patients with oral candidiasis can be treated with topical antifungal medications but most of these contain sugars which can exacerbate caries.[3,13]

Enamel hypoplasia and opacity defects

Drugs may cause enamel hypoplasia (deficient quantity of enamel resulting from developmental aberrations) and opacity defects. These include tetracyclines, thalidomide, fluoride and some authors include anti-neoplastic drugs used for chemotherapy[9,34,40]. The timing of drug administration is important as hypoplasia occurs during enamel matrix formation, which spans from the second trimester (when the primary incisors commence mineralisation) to about eighteen years of age (when the third molars erupt).[40]

Enamel hypoplasia can possibly increase the prevalence of caries post-eruptively (eg. within 6 months post-eruption) due to the discontinuous protective surface of enamel. It has been stated that these teeth become more prone to attrition and trauma (eg. defects on the incisal edges of primary teeth)[40]. Children on long-term medications that are sweetened, of low pH or that cause either xerostomia or enamel hypoplasia, are at higher caries risk of their teeth being "weak and soft".[3,11,12,18,26,27,31,38,40]

MYTH: "My child's teeth came out discoloured (black or brown)."

Tooth discolouration may be intrinsic or extrinsic and medicaments may cause both of these.

Extrinsic discolouration

Medicaments causing extrinsic discolourations are iron sulphide or iron supplements that stain the cervical margins of teeth black[6,16]. Stannous fluoride stains teeth brown.[16] Chlorhexidine mouthwashes if used long-term may cause black staining of teeth and tongue.[8,33]

Intrinsic discolouration

Intrinsic stains are caused by sulphur-containing drugs (black), fluoride (white) and tetracycline compounds.[6,16] One case report has postulated that

ciprofloxacin (a fluoroquinolone) therapy caused greenish discolouration when administered to infants (4 days to 1 month of age), but this requires confirmation of the association with ciprofloxacin.[25]

Tetracyclines cause several staining patterns depending on the analogue used.[6] Chlortetracycline produces a grayish-brown pigment whereas tetracycline, oxytetracycline and demethylchlortetracycline produce a brownish-yellow to yellow hue (yellow to brown, then darkens with exposure to light).[6,32] Discolouration of a developing dentition occurs when the drug is administered to pregnant women, nursing mothers or children under 12 years of age.[32] It has been recommended that tetracyclines should not be administered in the last trimester of pregnancy or children below the age of 6-8 years.[34]

Minocycline, a semisynthetic derivative of tetracycline, commonly used to treat facial acne and rheumatoid arthritis, causes teeth (blue gray to gray hue) and alveolar bone (black) discolouration. The oral mucosa appears pigmented due to the black pigmented alveolar bone showing through[6,32]. This occurs in about 2% of the population taking the drug for 2 months or longer. Unlike tetracycline staining, minocycline staining does not fluoresce; it can affect the dentition at any age, causes bone pigmentation and may regress with large doses of vitamin C or other antioxidants. The actual process of this unique staining remains an enigma.[6]

The evidence points to the fact that it may be true when parents claim that the teeth were discoloured (intrinsic) when erupted. However, some medications may cause extrinsic staining which occurs at any age depending on when and how long they are taken.

MYTH: "Drinking more milk builds stronger teeth".

Patients may believe that consuming more dairy products strengthens teeth as they are high in calcium. Milk provides energy, calcium and phosphate for the formation of hard tissues in growing infants.[46] It has a topical effect on the dentition as it can buffer the pH changes induced by acidic or fermentable substances.[36,46] New advances are being made with bovine milk protein casein phosphopeptide-amorphous calcium phosphate complexes (CPP-ACP) which exhibit anticariogenic properties and are being added to food, chewing gum, toothpaste and mouthwashes.[36,46] Thus there may actually be some truth in the myth that milk and its products (including CCP-ACP) strengthen teeth via a topical effect.

MYTH: "Is there any medication that I should have taken during pregnancy, or that I can give to my growing child now, to make his developing teeth stronger?"

Fluoride supplements

Parents often want to know if there is any "magic pill" they can take themselves or give their child so that their teeth will be "stronger". The use of fluoride supplements to prevent caries has been recognised for some time. However, with the lower prevalence of dental caries, water fluoridation and the diffusion effect to non-fluoridated areas and the widespread use of fluoride toothpastes, the benefits of supplements are being questioned.

There is evidence in the literature of both pre-eruptive and post-eruptive benefits of fluoride supplements but now their effectiveness is smaller due to the dilution and diffusion effect from other sources of fluoride exposure.[17] The benefits to the offspring of pregnant women who take supplements remain equivocal and it is impossible to ascertain if the benefits of supplements to newly erupted teeth are pre-eruptive or post-eruptive (topical effect).[17]

The pre-eruptive benefits of fluoride supplements are now being weighed against the risks of dental fluorosis.[24,37,47] A recent conference was held in Canada regarding the appropriate use of fluoride supplements.[24,37,47] Riordan concluded that supplements have caused dental fluorosis in many children and that there is poor evidence to support a caries-preventive effect.[37] For example, in a non-fluoridated area (Norway) with no fluoride diffusion effect, supplements provided no additional caries-preventive effect in 8 year olds even though the only other fluoride source was toothpaste.[47]

Limeback re-examined the pre-eruptive and post-eruptive mechanisms of the anti-caries effects of fluoride. He stated that ingesting fluoride supplements did not significantly improve the caries-reducing effects of fluoride as compared to fluoride rinse therapies.[24] Fluoride supplements given to pregnant women did not significantly improve the dental health of their child, possibly because fluoride levels in the fetal circulation were hardly affected by elevation in maternal blood supply. He concluded that near-maximum protection against caries is achieved without the need to swallow fluoride in the first three years of life and that liquid supplements could be administered to high-risk children (above 3 years).[24]

Authors unanimously agree on the topical post-eruptive effect of fluoride lozenges for high risk patients (school-age children and adults) after modifying the ADA and AAPD dosage schedules.[17,24,37,47] This implies that patient selection is important when prescribing fluoride supplements as not all children (and mothers) benefit from them and indiscriminate use may increase the risk of dental fluorosis.

MYTH: "Bad teeth due to unfluoridated water."

There is evidence to show that fluoridated water ranging from 0.7-1.2 ppm results in caries reduction.[5,24] These days, caries prevalence has decreased in areas with or without water fluoridation due to the presence of the "halo" effect and the more widespread use of fluoride toothpastes.[1,5] The apparent diminution of measurable benefits of community water fluoridation, results from the use of other fluoride-containing products that have contributed to lower levels of caries in both fluoridated and unfluoridated communities.[17,24] Therefore, it is not appropriate to use the lack of fluoride in the water as an excuse for caries formation as other fluoride sources are available such as fluoride toothpastes, fluoride mouthrinses and the presence of the "halo" or "diffusion" effect.[1]

Thus if parents ensure that their child is using a fluoride toothpaste twice daily and do not have a highly cariogenic diet, their caries prevalence

should not be any higher than those children in optimally fluoridated areas.

MYTH: "Placing aspirin on a tooth that is hurting will relieve a toothache."

Patients may place an aspirin tablet in the mucobuccal fold opposite a symptomatic tooth to relieve pain. Aspirin is one of the most extensively used drugs for pain relief in certain countries and has beneficial effects if used as instructed. However, inappropriate local application to the oral mucosa causes an aspirin burn or a painful, irregular-shaped ulcer.[21]

Realities

REALITY: Injudicious use of some beneficial medications can cause undesirable effects on oral health.

Examples of injudicious use of "beneficial" medications include chewing excessive vitamin C tablets (ascorbic acid) and iron preparations in a chewable form or iron tonics of low pH which may cause dental erosion if retained in the mouth.[26,42,49] Acetylsalicylic acid (aspirin) chewed daily on a prolonged basis for treatment of juvenile arthritis causes dental erosion.[26,49] In dental practice, many of the agents used are potentially harmful to the soft tissues such as formocresol, phosphoric acid, sodium perborate and hydrogen peroxide. If these agents accidently come into contact with the oral mucosa, erosive lesions or burns may occur.[33] Even though medications are essential for good health, they should be used properly in order to prevent any unwanted effects.

REALITY: Some medications can cause gingival overgrowth

Gingival overgrowth

Over fifty drugs may cause gingival overgrowth, however, the actual mechanism of this is poorly understood.[4,35] The drugs include anticonvulsants, calcium channel blocking agents, cyclosporin, and Rees et al speculate cannabis and erythromycin.[4,29,35] Gingival overgrowth in severe cases is unaesthetic, may interfere with speech and mastication and cause delayed tooth eruption.[22,29] Some authors claim that patients are at an increased risk for periodontal disease, ectopic eruption of teeth and halitosis.[4,22,35] The overgrowth usually begins in the interdental areas and the anterior labial regions are mostly affected.[4,29] Children (aged less than 6 years) appear to be more susceptible to gingival overgrowth than adults.[22,35,41,44,48]

Phenytoin causes a higher incidence of gingival overgrowth than other anticonvulsants.[29] Overgrowth begins to develop within a few weeks to months following initiation of phenytoin therapy, reaching maximum severity after twelve to eighteen months.[35] The prevalence is about 50% for patients and is higher in children, teenagers and institutionalised individuals.[4,29,35] Today, a number of alternative anticonvulsants are available which do not cause gingival overgrowth.[35]

It is possible in many cases (but not all) for the neurologist to discontinue the usage of phenytoin and to change to a different drug.

The calcium channel blocker commonly associated with gingival overgrowth is nifedipine. Incidence of overgrowth varies between 0.5% to 83% with an average of about 38%.[4,29,35] The presence is individual and dose dependant. It occurs within one to three months following drug initiation. Improvement does not always ensue following discontinuation of the drug and most other calcium channel blockers are associated with gingival overgrowth although to a lesser extent.[4,29,35]

Cyclosporin is an immunosuppressant drug that selectively affects cell-mediated immune response and is commonly used following organ and bone marrow transplantation to prevent graft rejection.[4,35,48] The prevalence of gingival overgrowth associated with the usage of this drug ranges from 2% to 85%.[22,29] Many patients taking cyclosporin are also on nifedipine to treat the cyclosporin-induced high blood pressure which may act synergistically on gingival overgrowth.[4,22,29,35,41,44] Gingival overgrowth occurs 1 to 3 months following initiation of the drug, reaching a plateau after a year.[29,35,43] Tacrolimus is a possible substitute which does not cause gingival overgrowth.[43]

Many studies implicate plaque in the pathogenesis of gingival overgrowth in organ transplant patients. However, better oral hygiene alone may not prevent the overgrowth or its recurrence in susceptible individuals.[22,41,48] Meticulous oral hygiene established before organ transplant may minimize symptoms.[4,29,35,43] Gingival overgrowth does not occur in all patients; nonetheless, good oral hygiene should still be recommended.[22]

REALITY: Medications used for oncology/transplant patients increase their susceptibility to infections.

Oral infections

Organ transplant patients are susceptible to fungal and viral opportunistic infections such as candidiasis and CMV infections.[13,41,43] This is because the patient is immunosuppressed, is on broad-spectrum antibiotics, steroid therapy and may have xerostomia.[9,13] Bone marrow transplant patients have a higher prevalence of candidiasis than solid organ transplant recipients (liver transplant patients have a higher prevalence of candidiasis than kidney or heart transplant patients).[8,41] Treatment of oral candidiasis is by topical antifungal agents and most of these contain sucrose or dextrose to mask the bad taste and improve compliance.[13] This may increase caries prevalence as these patients may also have xerostomia and may be on other sweetened medications.[9,13]

REALITY: Chemotherapy and radiotherapy affect oral health.

Chemotherapy and radiotherapy

Pediatric oncology patients on radiotherapy or chemotherapy have additional concerns as 90% of them will present with oral side effects.[7,8] These include mucositis, infections (fungal and viral), xerostomia, taste disturbances, non-gingival soft tissue overgrowths, dental hypersensitivity, tooth agenesis, complete or partially arrest of root development, dentine and enamel opacities and defects (due to hyperpyrexia during neutropenic phases), microdontia, delayed eruption

of permanent teeth and reduction in lower face height due to growth disturbance of the maxilla. Children on growth hormone therapy may also have relatively increased mandibular growth.[7,8,9]

These patients have a higher chance of developing dental caries due to xerostomia, reduced salivary flow rates, lowered buffering capacity of saliva and higher amounts of cariogenic bacteria found post-transplant (BMT). According to Dens et al these factors when coupled with a soft cariogenic diet during the acute phase of treatment, may lead to irradiation caries.[7,10]

REALITY: Chlorhexidine mouthwashes are effective in relieving mucositis.

Chlorhexidine

Chlorhexidine is a widely used and effective disinfectant in the oral cavity and has been recommended for oncology and transplant patients as a preventive measure against infections.[10,13,33,43]

However, a few recent reports speculate that chlorhexidine mouthwashes are ineffective in lowering the incidence of mucositis in oncology patients.[7,45] This fact coupled with the side-effects of chlorhexidine such as discolouration (extrinsic brown staining) of the teeth and tongue, taste disturbances, burning sensation in the mouth (alcohol based preparations), transient swelling of the parotid glands and reduced efficacy of other drugs, have alerted some authors to restrict its use only in patients with poor oral hygiene or periodontal problems.[7,8,33,45]

There is conflicting evidence for the use of chlorhexidine in oncology and transplant patients. Since many of the side-effects are not seen in the pediatric population and alcohol-free preparations are available, until more concrete evidence is given against its use, its use should not be discontinued.

Recommendations

Myths should be clarified by the dentist and patient education is important, as it is the dentist who has first hand information on what the parent or child believes or is taught to believe. Doctors, pharmacists and other health professionals need information so that they can either refer the patient to a dentist or advise patients appropriately on potential oral effects of medications and on fluoride supplements. Where possible, sugar-free liquid medications should be prescribed and pharmaceutical companies should be encouraged to manufacture more sugar-free pediatric preparations. On a community level, oral health campaigns and information disseminated via the media or schools can make people more aware of the "truth" behind the myths and allow them to make the right decisions.

The realities addressed are factual but may be unrecognised by health professionals and parents alike. All those involved with the medically compromised child (eg. pediatric dentists, doctors, nurses, specialists, dieticians, pharmacists, parents and caretakers) should be educated on the possible deleterious oral effects that may occur in association with medications. Protocols should be set up in hospitals such that all chronically sick, oncology and transplant pediatric patients receive a pre-operative dental examination and be followed up to maintain a comprehensive preventive regime. Children on long-term medications with known oral side effects should be seen regularly by a pediatric dentist.

Conclusion

Numerous statements about the effects of medications on dental health are presented in the literature or made by parents. Some are supportable (eg medications causing caries and discolourations, milk being anticariogenic) while others are not (eg. aspirin to relieve toothaches, benefits of fluoride supplements and bad teeth due to unfluoridated water). Some myths are now historical and these include tetracycline staining and aspirin burns. Current issues include the fact that certain medications predispose patients to dental caries, the "halo" effect of fluoride, the injudicious use of fluoride supplements and lastly the fact that milk is anticariogenic. Pediatric dentists must know the oral side effects of medications that are now used for longer-surviving oncology and transplant patients. Thus, we as professionals will be equipped with the knowledge and confidence to advise our patients accordingly.

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Postcards from Bergen

4TH CONGRESS , EUROPEAN ACADEMY OF PAEDIATRIC DENTISTRY, JUNE 2000

The Australian contingent. Taken at the opening ceremony of the 4th Congress of the European Academy of Paediatric Dentistry, Bergen, Norway in June 2000.

Pictured (*left to right*):

Back row: Roger Hall (Vic), Peter King (NSW), Erin Mahoney (NSW), Angus Cameron (NSW).

Front row: Vera Hall (Vic), Paul Riordan (WA), Katherine Ngu (NSW), Richard Widmer (NSW)

Lunch in Bergen

Pictured (*left to right*):

Rodney Studd, Erin Mahoney, Katherine Ngu, Eduardo Alcaino, Kay Hood, Peter King, Angus Cameron

Pictures submitted by Dr Angus Cameron

Attention Deficit Hyperactivity Disorder

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Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is a common developmental disorder affecting about 3-5% of the population (depending on the precise definition used). The term ADHD is currently used to describe a range of children with varying functional difficulties, who share the feature of poor sustained attention. Some are extremely impulsive, some aggressive, others quiet and restless whilst many have low self-esteem. Commonly associated problems (comorbidities) include developmental language disorders, anxiety, oppositional-defiant behaviours, fine motor and coordination difficulties and specific learning disabilities. Virtually all children with ADHD have deficits in short-term auditory memory, meaning they find it very difficult to retain more than one or two brief instructions in their minds.

Aetiology

The aetiology of ADHD is complex and while there is not currently (and is not likely to be) a diagnostic laboratory test for ADHD, research is continuing to identify some of the biological correlates of ADHD behaviours which then may prove helpful in refining more targeted therapies. Recently there has been a focus on neurochemical, electroencephalic, dynamic metabolic imaging and genetic differences between children with ADHD and controls. Levels of dopamine in the cerebrospinal fluid are lower in children with ADHD than controls. On standard electroencephalogram (EEG) children with ADHD have been found to have increased theta (4-8 Hz, particularly frontally) and decreased beta 1 discharges (12-20 Hz, particularly temporally) compared to controls, which is accentuated under task conditions e.g. reading, drawing. Single-photon emission computed tomograph (SPECT) studies, which examine cerebral blood flow, have demonstrated relative hypoperfusion of the frontal and striatal regions in subjects with ADHD compared to controls. Strong evidence for a genetic contribution has emerged from Australian twin studies, demonstrating much higher concordance rated among pairs of monozygotic twins than same-sex dizygotic twins. In summary, ADHD appears to be a biologically determined condition, the manifestations of which are modified by environmental circumstances.

Current theory regarding the neuropsychological basis of the observed behaviours in children with ADHD centres on the concept of response inhibition. These children have deficits in self-regulation. They seem to be less able to inhibit cognitive +/- motor impulses than most children. They have a reduced capacity for “working memory”. This can be thought of as like the RAM of a computer. That is they are not good at retaining information for use in the next stage of a task. In addition their internalisation of language is less well developed than that of their same age peers. These factors mean that children with ADHD find it very hard to plan and persist with tasks and activities directed toward a goal that is less than immediate.

The above difficulties with so-called “executive function” translate into the core functional difficulties manifested by children with ADHD. These include poor effort persistence, inability to tolerate delayed gratification of needs and unpredictable outward expressions of impulses (eg. talking, moving). There is often excessive motor activity.

Diagnosis

The assessment of a child for the diagnosis of ADHD requires a number of essential components. These include a detailed developmental history, physical, neurological and neurodevelopmental examination, and obtaining detailed standardised behaviour rating scale data from at least two sources, usually school and home. A number of instruments are available. This is scored to determine whether the reported symptoms are statistically deviant relative to normative community data. Many clinicians and most researchers in Australia and North America use the diagnostic criteria of the Diagnostic and Statistical Manual of Mental Disorders 4th Edition - DSM IV. This stipulates that a diagnosis of ADHD can only be made if the symptoms have been present since below age 7, are observed in at least 2 settings (ie. school and home) and have persisted for at least 6 months (to exclude adjustment reactions to environmental stressors such as parental separation, change of school, death of grandparent etc). In addition the behaviour exhibited by the child must be maladaptive (ie causing social, academic or functional impairment) and be present to a developmentally inappropriate degree. All 2-year olds and most 3-year olds and many 4- and 5-year olds are impulsive and inattentive, so clearly the child's behaviour must be evaluated relative to age-related standards for meaningful interpretation. Finally in order for the symptoms (behaviours) to be ascribed to ADHD other psychiatric disorders such as autism and psychosis need to be excluded.

Children with unrecognised or untreated ADHD are at significantly increased risk of a range of negative developmental outcomes. These include academic failure, school dropout, delinquency, unemployment, relationship difficulties, injuries, substance abuse, criminal activity and incarceration. It is believed that early identification of children with ADHD, with early institution of multi-modal behavioural, academic +/- pharmacological therapies will improve the long-term prognosis. However, this is yet to be conclusively demonstrated in the few good quality longitudinal studies published.

Management

Management of the child with ADHD involves three broad approaches; behavioural, educational and pharmacological. Many other approaches are commonly applied to these children, including dietary modification, “natural” or

complimentary therapies of diverse types, and behavioural optometry. There is little evidence to support the broad use of any of these interventions, though some individuals report benefits.

Stimulant medication is the principal pharmacological therapy for ADHD. Other medications sometimes used in ADHD include the anti-hypertensive clonidine (Catapres), anti-depressants (selective serotonin re-uptake inhibitors, reversible monoamine oxidase inhibitors, tricyclics) and occasionally major tranquillisers.

Stimulants are believed to work by increasing the amount of catecholamine neurotransmitter in the synaptic cleft, either by increasing the amount of stored neurotransmitter (dopamine) released from the presynaptic neurone or by blocking the post synaptic uptake of the neurotransmitter. This increases activity particularly in the prefrontal cortex and limbic system, areas associated with attention and arousal.

The two stimulants available in Australia are dexamphetamine and methyl-phenidate (Ritalin). Despite having a greater risk of side effects, dexamphetamine is prescribed more widely than Ritalin in Australia. This may relate to cost differential – dexamphetamine is a PBS item, whereas Ritalin is not and costs between $48 and $80 for 100 tablets (about a month's supply). The prescription of stimulant medications in Australia is restricted to paediatricians and child psychiatrists.

A comprehensive approach to the management of a child with ADHD often involves medication in conjunction with educational and behavioural strategies. The aims of any educational approach is to maximise attention span, try to control impulsive behaviour, assist with learning difficulties and thus raise self esteem. Such programmes may include specific skills training, role-play exercises and the appropriate use of educational aides. Any educational programme is more likely to be successful if supported by behavioural strategies which help promote socially appropriate behaviour eg. training a child to raise their hand when wanting to speak, appropriate consequences for inappropriate behaviour eg 'time out' etc. In addition programmes to assist the child in managing anger and frustration and family supports may also be useful.

The dental environment

The visit to a dentist is likely to raise anxiety levels in any child and indeed their parents. In a child with ADHD this anxiety may manifest in overexcited behaviour. Many parents worry about the affect of their child's behaviour on others. They have become accustomed to failure having taken their 'difficult' child to dentists only to be told that it is not possible to provide treatment/care. The result is either an excessively protective/embarrassed parent with constant apologies on behalf of the child or a somewhat aggressive parent exerting inappropriate, heavy handed disciplinary actions throughout the encounter. In either situation the child's behaviour is likely to be reactive towards the parent thus precluding the establishment of a successful relationship with the dental practitioner.

In general the chance of success is raised if the dental practitioner takes control of the situation early. This can be done by being quietly firm with both the child and the parent. By creating an atmosphere of confidence the parental anxiety is often alleviated allowing the child and the dentist to establish a relationship in a more relaxed environment. Likewise a gentle but firm approach will convey to the child a confidence and a structure to the situation within which it is easier for them to conform.

In the dental environment the inability to sit still coupled with the impulsive and unpredictable behaviour can make even a simple examination challenging let alone more complex dental treatment. It is useful for the dental practitioner to have an understanding of the current management strategies being employed by the family at home and in school. For example, if a child is used to raising their hand prior to speaking, it is useful for the dentist to employ the same strategy. The current medication scheme should be discussed with both the parents and the prescribing practitioner. It is often possible to modify either the dose or the timing of medication to optimise the action at the time of the dental visit.

Clear instructions should be given to the child maintaining eye contact throughout and taking care not to over burden the short-term memory. Such instructions need to be given at a time when the child is not distracted by other activities in the dental surgery eg. the chairside assistant removing instruments or setting up for the next patient.

It goes without saying that prevention is essential for these children. Minimising the need for complex restorative treatment will undoubtedly make life simpler. However, it is again important to realise that many of these children are already struggling to master other life skills. Brushing their teeth or controlling their diet both require concentration, motivation and understanding all of which can be problematic for the child with ADHD. Toothbrushing charts for the child to take home and mark off daily are more likely be successful than verbal instructions to brush daily. Repetition is important in building up self-confidence in the child. Multiple short visits have a higher chance of success than single prolonged ones. However, it is important to realize that oral health is only one of many priorities that the child with ADHD has and the multiple demands made of the parents needs to be weighed against the overall needs.

Praise and encouragement play an important role in the management of these children and good behaviour should be reinforced and rewarded. On the other hand, knowledge of the behaviour management strategies that the child is familiar with may also assist in allowing the dentist to take control of the sessions in order to optimise the outcomes.

Conclusions

ADHD presents a clinical challenge for oral health practitioners. Whether ADHD is increasing is unclear. However, our understanding of the aetiology and pathophysiology is developing rapidly. The management of children with this disorder is complex and multifaceted. It is likely that dentists will come across these children increasingly and an understanding of the condition and its management is essential if we are to be successful in promoting good oral health.

Serial Extraction Revisited

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Introduction and history of serial extraction

Serial extraction can be defined as the appropriately timed, planned removal of selected primary and permanent teeth in a developing dentition with severe tooth-size to jaw size discrepancy so as to improve the alignment of permanent teeth as they emerge into the oral cavity.

In 1743, two Frenchmen, Bunon (a pediatric dentist) and Bourdet, were the first to recommend extraction of teeth so as to relieve crowding. Later, in 1851, Linderer from Germany wrote about extraction of teeth to relieve crowding in his book[14]. The term "serial extraction" was introduced by Kjellgren (a Norwegian) in 1929, and Hotz (a Swiss) in 1948 used the term "guidance of eruption" to describe the same procedure[3,14]. Some authors prefer to use the term guidance of eruption because the development of occlusion is the main concern not the extraction of teeth per se[2,3,14]. In 1949, Heath in Australia (Melbourne) described four procedures which he referred to as "planned minimum orthodontic treatment", which is carried out when a malocclusion is inevitable. This is basically a modification of the serial extraction procedures that are carried out today[13].

Later, American authors Dewel (1954), Tweed (1966) used the "term preorthodontic guidance", while Mayne (1969), Graber (1972) and Dale (1985) wrote further about serial extraction[10,15]. As advances in the study of growth and development of the jaws were made, these authors stressed the importance of diagnosis and case selection in serial extraction cases.

Serial extraction is not a straightforward procedure. The clinician needs to understand the transition from the primary to the permanent dentition, how much potential growth can occur during the mixed dentition period, and also the timing and sequence of extractions[2,10]. However, as easy as it may sound, it is essential that the clinician undertaking this procedure have a good knowledge of proper diagnosis and case selection, the indications and contraindications, the actual extraction sequences and the advantages and disadvantages of the procedure.

Diagnosis

Case selection is the most crucial factor for serial extraction. The orofacial area is composed of three important tissue systems: namely the dental, skeletal and neuromuscular systems. When considering serial extractions, the clinician has to assess the inter-relationship between these three systems[12,21].

Dental development

In the mixed dentition, mild crowding is observed usually after eruption of the mandibular incisors. This is due to the fact that permanent anterior teeth are wider mesiodistally than their primary predecessors. However, nature has a carefully conceived plan to accommodate the wider permanent teeth so as to allow harmonious physiologic succession as the primary dentition progresses to the permanent dentition[17,21].

Incisor liability

This term was coined by Mayne to define the discrepancy in widths between the four permanent incisors and their primary counterparts[2,21]. The four maxillary permanent incisors are 7.6mm larger than the deciduous incisors on average and the value for the mandibular teeth is about 6.0mm[2,12,21]. There are four factors that will occur either singly or in combination that can naturally compensate for this difference[2,21]. They include the interdental spacing of the primary incisor teeth, the intercanine arch width growth, the increase in intercanine arch length and the favourable variations in the size ratio between the permanent and primary teeth.

Interdental spacing of the primary incisor teeth

This may range from 0-10mm in the maxilla (mean 4mm) and 0-6mm in the mandible (mean 3mm).

Primary spacing refers to the spacing already present in the primary dentition. Secondary spacing occurs in a "closed" dentition (without primary spacing) as the intercanine arch width increases during the period of emergence of the permanent incisors. Kluemper et al[17] have added the primate space as a compensatory factor as well.

Intercanine arch width growth

This is the most unpredictable and diagnostically challenging variable of the developing dentition. In the mandible it occurs between age 6-9 years in boys and age 6-8 years for girls, and in the maxilla it increases until about 16 years in boys and 12 years in girls[22]. This increase is about 3mm in the mandible and 4.5mm in the maxilla. After 10 years, there is little change in arch width in both sexes[22].

Intercanine arch length increase through labial positioning of the erupting permanent incisors

The permanent incisor teeth erupt in a more procumbant position than the primary incisor teeth. In 1950, Baume estimated that this can provide up to about 2.2mm in the maxilla and 1.3mm in the mandible. However, this may be transient as the incisors may upright themselves with time[2].

Favourable variations in the size ratio between the permanent and primary teeth

This refers to the amount of leeway space present. The greater the leeway space, the less space will be required from the anterior segments to accommodate the posterior teeth.

An impossible situation arises when there is a true hereditary tooth-size/jaw-size discrepancy present together with an incisor liability that cannot be compensated by the above mentioned factors. It is in these cases that serial extraction can be beneficial.

Leeway space

Nance described the size difference between the primary canine and molars and the permanent canine and premolars as leeway space.

This difference is about 2.6mm in the maxilla and 6.2mm in the mandible and varies considerably between individuals[2]. However, the mandibular leeway space is more apparent than real[15,17,21,22]. Mesial drifting of the mandibular first molars takes up most of the leeway space so as to allow a Class one molar relationship to be established[12,15,17,21]. This can occur either when the first molars first erupt (early mesial shift) in a spaced primary dentition, or after exfoliation of the mandibular second molars in a closed primary dentition (late mesial shift)[2].

There is evidence to show that posterior teeth move mesially throughout life. Therefore, many authors have concluded that after eruption of the first molars, arch length does not increase but may actually decrease[1,2,12,21]. Serial extraction is based on this fact and thus if crowding is apparent by about 8 years of age, the crowding will have little chance to improve with further growth and development[2].

Skeletal development

The functional articulation between the basal bones to each other and their relationship to the cranial base has to be within normal limits in a case selected for serial extraction. Thus cephalometric analyses are required for all cases that can potentially be treated by serial extraction. Skeletal maturity is also important and a hand wrist radiograph could be used. This will enable the clinician to predict remaining growth and anticipated developmental adjustments of dentofacial relationships during growth.

Muscular development

When serial extraction is under consideration, the perioral musculature should not be overlooked. Imbalances in musculature should be noted such as strain, hypotonicity, hypertonicity, unusual lip lines or markings. Any adverse oral habits should also be noted such as thumb sucking. Serial extractions should only be carried out in cases where there is normal orofacial musculature and no adverse oral habits are detected[21].

Rationale for serial extraction

The rationale for serial extraction is that first, it is possible to predict with a fair degree of certainty that there will not be enough space for the permanent teeth to erupt[1,12,15,28]. Second, after the eruption of the first molars, there is no increase in arch length and also iatrogenically produced increased arch length by expansion is not very stable[1,12]. Lastly, early removal of teeth will allow for a more physiologic unassisted movement of adjacent teeth into more favourable positions[1,17].

Indications for serial extraction

A synopsis of the literature states that the clear indications for serial extraction cases are as follows[2]:

1. A true, hereditary, relatively severe jaw-size/tooth-size discrepancy (about 8-10mm)[15,23]
   - Maxillary mandibular alveo-lodental protrusion without spacing
   - Crowded mandibular incisor teeth
   - A midline displacement of mandibular incisor teeth due to premature exfoliation of primary canine on the crowded side
   - Mandibular incisor blocked out lingually or occasionally labially or impacted
   - Crowded mandibular lateral incisors that have commenced resorbing the roots of the primary canines
   - Bilateral premature loss of a primary canine leading to upright positioning of incisors, thus increasing the overjet and/or overbite
   - Splaying of incisor teeth due to crowded positioning of unerupted permanent canines
   - Gingival recession and alveolar destruction of the labial surface of the prominent mandibular incisor[8]
   - Ectopic eruption of the permanent maxillary first molar indicating a lack of development in the tuberosity area
   - Radiographically demonstrated vertical palisading of the permanent first, second and third molars indicating a lack of jaw development
2. A mesial step mixed dentition developing into a Class one molar relationship
3. A minimal overjet relationship of the incisor teeth
4. Minimal overbite
5. A facial pattern that is orthognathic or with a slight alveolodental protrusion (bimaxillary protrusion)
6. A harmonious Class one antero-posterior relationship of the maxilla and the mandible to each other[12,21].
7. The facial profile can be described as flat or straight[8].
8. Radiographic and/or clinical evidence of all permanent teeth in good condition[11]
9. Mesial drift of buccal segments[12], and
10. A commitment on the practitioner's part to completing the case once commenced[23].

Contraindications for serial extraction

A synopsis of the literature indicates that these are the contraindications for serial extraction:

1. Crowding caused by environmental factors or when crowding is slight and there is not a substantial lack of space[2,11]
2. Increased overjet or reverse overjet[15]
3. Deep overbite or an open bite. These should be treated before undertaking serial extraction[11]
4. Permanent teeth congenitally missing from the dental arch[11]
5. Gross malposition of teeth, rotations and crossbites[15]
6. Marked skeletal discrepancy in the arches[11,24]

Borderline cases

These include cases where mild incisor crowding is present which can be relieved by proximal stripping of the deciduous canines[14]. In cases where there is a favourable size ratio of the buccal segments, but with crowding of the incisors, early expansion may be used[14]. In cases where incisors are well-aligned and there is an unfavourable size ratio of the buccal segments, serial extraction should not be carried out[14]. In Class two cases with marked crowding in the mandible, serial extraction or guided eruption procedures should not be carried out

and functional appliances could be used instead[14].

Borderline cases will have good facial patterns, moderate loss of arch length, good muscular environment and a satisfactory direction of skeletal growth[7,8]. In mild borderline cases, there is no urgency for extraction and growth should be monitored; the patient can either have orthodontic treatment without extractions or if growth is inadequate, a traditional serial extraction sequence or an alternative sequence to serial extraction can still be carried out[7,9]. When in doubt after a mixed dentition analysis, it is always wise to consider the advantages of postponing treatment until complete eruption of the permanent dentition[9].

Serial extraction procedure

There are three factors that should be considered by the clinician to assist in deciding which teeth to extract and when to extract them. These include the effect of extraction of the primary tooth on the eruption rate of its permanent successor, the amount of root formation at time of emergence, and the length of time for the attainment of various stages of root development[2,22].

It is important to stress at this point that serial extraction is a long-term guidance program and it may be necessary to re-evaluate and modify earlier decisions as treatment progresses[12]. The objectives of serial extraction are to make treatment easier and later mechanotherapy less complicated, less expensive and shorter[2].

The classic procedure described in the literature is the removal of the primary canines followed by the primary molars, and finally extraction of the permanent first premolar[2,6,12]. However, the procedure has become increasingly sophisticated and precise, and the sequence is varied according to the diagnosis. Some examples of the different sequences for different situations will be described below[2]:

Serial extraction in Class I treatment

Anterior discrepancy: crowding (primary canine, primary first molar, permanent first premolar)

The first extraction is of the primary canines, to relieve incisor crowding after eruption of the lateral incisor. Then extraction of the first primary molar is performed after incisor crowding has improved and the extraction site is reduced in size. First premolars should have half root formation at this point as this theoretically enables them to erupt earlier, ahead of the canines.

When the permanent canines have developed beyond one half root length, indicating an acceleration in their eruption, the first premolars are extracted (this happens more frequently in the maxillary arch as in the mandibular arch, the first premolars erupt after the canines). This is to allow distal drift of the erupting permanent canines[12].

Anterior discrepancy: alveolodental protrusion (primary first molar, primary canine and permanent first premolar)

Extraction of the primary first molars is the first step because only a minor irregularity of the incisor teeth will be present. Premolars have to be at half root formation in order to encourage their early eruption ahead of the canines. However, if the primary first molar is extracted too early, this may cause delayed eruption of the premolar and an undesirable knife-edged ridge. Next the primary canines and first premolars are extracted so as to encourage lingual tipping of the incisors.

Middle discrepancy: impacted canines (primary first molar, permanent first premolar)

In this situation, there may already be premature exfoliation of the primary canines. The incisors may be splayed out due to crowding in the apical region. The first deciduous molars should be removed to encourage the premolars to erupt early (at about half root development).

The premolars are then extracted so that the impacted permanent maxillary canine will have space to migrate away from the apices of the lateral incisors. Every effort should be made to avoid correcting the incisors with fixed appliances for fear of incisor root resorption. The greater concern is for the canines to correct themselves and not incisor irregularity.

Tooth germ enucleation in the mandible (primary first molar and permanent first premolar)

If the canines appear to be erupting before the first premolars, then extraction of the primary first molars with subsequent enucleation of the first premolars is indicated. This will allow the distal migration of the erupting canines.

In the maxilla however, this is not a usual occurrence so enucleation is less likely to be indicated. The sequence can be modified if there is no incisor crowding such that first primary molars are extracted and then the primary canines together with the first premolars can be removed.

Tooth germ enucleation in the maxilla and mandible (primary canine, primary first molar and permanent first premolar)

On rare occasions in both the maxilla and mandible, the permanent canines will erupt before the premolars. Thus the sequence will have to be extraction of the primary canines followed by the first molars and enucleate the first premolars. This is only recommended if fixed appliances are not a possibility.

Alternative to tooth germ enucleation (primary first molar, primary second molar, permanent first premolar)

Enucleation should be avoided if an opportunity exists to place fixed appliances at the completion of serial extraction. When the permanent canines are erupting ahead of the first premolars, then first primary molars should be removed following half root formation of the first premolars.

Some 6 to 9 months later, when the erupting first premolar appears to be obstructed by the second deciduous molar, the deciduous second molar should be extracted. This is usually not necessary in the maxillary arch and the maxillary primary canine and first premolar should be extracted.

When the mandibular first premolars have erupted, then they should be extracted. Removal of the mandibular second primary molar usually increases the likelihood that a lower lingual holding arch may be needed to prevent space loss due to the mesial drifting of the first permanent molars[12].

Advantages of serial extraction

Serial extraction reduces appliance treatment time, the cost of treatment, discomfort to patients and time lost by both parents and patients[3]. It intercepts the developing malocclusion as early as possible so as to reduce, or in rare instances avoid, orthodontic treatment at the sensitive teenage period[3]. Serial extraction is aimed at encouraging a measure of self-correction in order to shorten the time and complexity of mechan-otherapy[1,6,21,28]. If the perio-dontium was affected originally, the gingival health of the primary incisors will benefit from this procedure[1,20,21]. Psychologically, the child will benefit from earlier correction of aesthetics as the anterior teeth spontaneously align themselves[1,20,21]. Some authors claim that retention requirements in serial extraction cases are markedly lessened[2,21]. There will also be less potential for iatrogenic orthodontic damage to tooth roots if serial extraction is carried out, because the orthodontic treatment time is reduced[21]. Serial extraction gives the erupting teeth the chance to become aligned as they erupt into the oral cavity instead of staying in a crowded unfavourable condition for many years[2,28].

Yoshihara et al. have stated that it may be possible (though not ideal) for serial extraction to be used as the sole form of treatment[28]. This depends on the financial situation of the parent and also on the decision of the parent/patient and practitioner that no active treatment is warranted. A further point raised in the paper was that under appropriate circumstances, serial extraction could be used on handicapped patients who are unable or unwilling to accept orthodontic therapy[28].

Disadvantages of serial extraction

The disadvantages of serial extraction include an increase in overbite, lingual tipping of the mandibular incisors thereby decreasing arch length, and fixed appliance therapy after a long period of follow up[1]. Early extractions can also lead to space loss, delayed eruption of the permanent successor, and elimination of the opportunity to extract other teeth that may become doubtful such as the first permanent molars. Once permanent teeth are extracted, the patient is committed to fixed appliance therapy[15].

An important consideration, which may be overlooked by many clinicians, is the fact that the child will have to be subjected to multiple extractions (up to sixteen in some cases) under either local or general anaesthesia. Since the Poswillo report in 1992, there has been a greater effort to reduce the use of general anaesthesia for orthodontic extractions in children in the United Kingdom[26,27]. Shaw and Shaw have suggested the use of inhalation sedation together with local anaesthetic to perform an extraction instead of a general anaesthesia as it is clinically more successful, cost-effective and better accepted by children and their parents[26,27].

A study on the post-retention stability and relapse of 30 serial extraction cases (after 10 years) by Little et al demonstrated no difference between cases treated with or without serial extraction after fixed appliance therapy[19]. There will always be some irregularity present for the majority of cases in which retention is discontinued. Therefore they stress the importance of life-long periodic use of retention[19].

Lee followed up seven cases where he monitored the behaviour of erupting crowded lower incisors for about 13 years and found that the crowding resolved spontaneously in this small group of patients[18]. He argued that the extraction of primary canines in serial extraction is questionable and that primary canines are important for the optimal development of the mandibular intercanine area[18].

Discussion

Serial extraction sequences involving the second permanent premolars are indicated in two situations. Firstly is when there is an impaction of the second premolars in cases where there is loss of arch length due to premature loss of deciduous second molars[16]. The second situation occurs when there is minimal crowding in the anterior portion of the mandibular arch or there is a posterior arch length deficiency or if the clinician wishes to reduce the possibility of facial profile flattening[16]. The disadvantages of second premolar extractions are that early decisions are required to be made and also that enucleation of the premolars is required; if bone is removed injudiciously, this will lead to an undesirable knife-edge ridge formation[16]. Other important points to consider when extracting second premolars are the restorative status of the premolar segments, tooth shape and the amount of overjet and overbite present[12]. If there is an open bite tendency, then second premolar removal in the mandibular arch is recommended as this reduces the tendency to relapse into an open bite and prevents the incisors from tipping lingually[12].

When considering timing of extraction of the first premolars, Jacobs found that some clinicians waited until the permanent canine is just about to erupt before extracting the first premolars as the first premolar is the best space maintainer and also because the canine may erupt much later than the first premolars[15]. This is more important in the maxillary arch than the mandibular arch as the premolars erupt before the canine in the maxillary arch and also because buccal segment drift is more marked in the maxilla[15].

Proffit et al, Dewel and Mayne do not advocate enucleation of the first premolars as it is a more traumatic procedure, leads to greater bone loss and eventually gives rise to poorer bony contours[5,21,24]. Serial extraction can be carried out in certain Class two malocclusions but if not done with extreme caution can actually aggravate the preexisting malocclusion[2].

In most instances, serial extraction procedures are instituted when the patient is about 8 years old. The patient is then followed up every 3-6 months as there is an interval of about 6-12 months between steps. Clinical and radiographic evidence of growth and development, sequence of eruption and self-adjustments have to be assessed so that the most favourable sequence can be advocated[12].

Serial extraction in itself will not ensure good aesthetics and function, although in a few cases it will be fully corrective. The great majority of the cases will require comprehensive orthodontic treatment. The treatment objectives of mechanotherapy after serial extraction are to: close residual spaces; improve the axial inclination of teeth; correct rotations; correct any

midline discrepancy; correct residual overjet and overbite; correct crossbites; refine the intercuspation of the dentition; and improve and coordinate arch form[2].

A precise arch length analysis is an essential part of every serial extraction diagnosis to determine the extent of space loss mesial to the first permanent molars[8]. Two other important assessments that must be made, are whether the molars can be moved distally, and if the incisors can be moved labially. Molars can only be moved distally if spacing exists between the first and second molars. Incisor inclination has to be assessed to see if the incisors can be moved labially to create space. In true serial extraction cases, the incisors are already labially placed[8].

Conclusion

Serial extraction is a procedure that needs to be carried out with extreme caution and deliberation by a trained clinician, such as a paediatric dentist or an orthodontist, who is committed to completing the case once initiated[5,25]. The procedure has been advocated for more than 200 years but is definitely not as simple as it is often perceived to be. Case selection and diagnosis are of utmost importance. The clinician and patient will have to be committed to a long-term period of observation, proper timing of an appropriate sequence of extractions, and finally to a period of fixed appliance therapy. Thus serial extraction should only be undertaken by a competent clinician who is committed to complete the case, or in special circumstances, where the procedure is the only viable option for a patient for whom fixed appliance therapy is not an option.

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Colgate Australia/New Zealand Oral Health Promotion Conference

Dr Jackie Robinson, Professional Relations Manager, Colgate Oral Care

The Australia/New Zealand Oral Health Promotion Conference was hosted by Colgate on 8 - 9 March at the State Library in Sydney. This was the third such conference held in Australia and is the only national forum dedicated specifically to oral health promotion. 140 delegates representing New Zealand and every state and territory of Australia attended the conference.

Above: Guest international speaker, Dr Richard Watt, University College London

The conference gave delegates the opportunity to hear about new knowledge shaping oral health promotion and facilitated communication amongst individuals driving oral health promotion projects across Australia and New Zealand. Like so many other aspects of oral health, concepts underlying oral health promotion have change significantly over the past two decades. New knowledge on effectively influencing positive health behaviour has moved oral health promotion beyond an emphasis on education alone. The new approach focuses on environmental factors as well as on individual behaviour and on access to professional advice and care. Increasingly oral health promotion will be linked with other synergistic health promotion campaigns.

The diverse two day conference program featured presentations from all states of Australia and from New Zealand. Dr. Richard Watt of University College London was the invited international guest speaker.

Dr. Watt has lectured and published widely on many facets of oral health promotion. His presentations at the conference centred on the common risk factor approach to oral health promotion and smoking cessation in the dental practice setting.

Leading figures in oral health promotion within Australia comprised the majority of the program. Dr. John Rogers of Melbourne and Ms. Karmen Hellmuth of Brisbane presented overviews of oral health promotion projects in their respective states. Prof. Clive Wright of Melbourne and Dr. Jane Chalmers of Adelaide spoke on programs and issues relating to the ageing population. Ms. Julie Satur of Melbourne addressed the questions of evidence based oral health promotion. Dr. Marc Tennant updated the audience on changes to the oral health system in Western Australia.

Indigenous health issues formed the basis for presentations by Ms. Lesley Steele of Adelaide and A/Prof. John Broughton of New Zealand. Dr. Peter King and Dr. Emma Jay of Sydney spoke on oral health issues associated with mental illness. Other outstanding presentations included "Cool Canteens", the Teethsmart program in South Australia and the "Bright Smiles, Bright Futures" schools program.

Above: Dr Antonia Scott (right), member of the ADA Oral Health Education Committee with Ms Marion James from Queensland

Above: Delegates atttending the Conference

The next Colgate Australia/New Zealand Oral Health Promotion Conference will be held in 2003.

Above: Guest speakers and Colgate hosts at the conference (L-R): Clive Wright (Vic), Lenore Tuckerman (Colgate), Peter King (NSW), Jane Chalmers (SA), Richard Watt (UK), Jackie Robinson (Colgate), Mark Tennant (WA), John Rogers (Vic), Julie Satur (Vic), Alison Miles (ACT), Lesley Steele (SA), Carol Nicholson (Tas), Karmen Hellmuth (Qld), Anne Hill (Vic), Susanne Sofranoff (Vic), Sue Elliott (SA), Christine Gardner (SA), John Broughton (NZ), Brian Howard (Colgate)

Coming events

- 7th World Congress on Preventive Dentistry. "Prevention in the 21st Century." Beijing, China. 24-27 April, 2001. Secretariat Office National Committee for Oral Health. 38 Baishiqiao Road, Haidian. Beijing, 100081, China.

  Congress Web Site:
  http://www.ciccst.org.cn/wcpd

- Australasian Academy of Paediatric Dentistry. Pre-Congress Meeting. Brisbane, Australia. 3-4 May, 2001.

- 30th Australian Dental Congress. Brisbane, Australia. 4-8 May, 2001. Contact Congress Secretariat, PO Box 1280 Milton, Qld. 4064

  e-mail: ada2001@im.com.au

- 9th International Congress on Cleft Palate and Related Craniofacial Anomalies. Göteborg, Sweden, 24-28 June 2001. Contact Conference Secretariat, Congrex Göteborg AB, Box 5078, SE 402 22 GÖTEBORG, Sweden.

- I.A.P.D. Congress, Paris, France. 13 - 15 September 2001.

- 89th FDI Annual World Dental Congress. Kuala Lumpur, Malaysia. 16-19 September, 2001. Contact Mr Paul Wilson, FDI World Dental Federation Congress & Exhibition, 7 Carlisle Street, London, England W1V 5RG.

- 13th Australian and New Zealand Biennial Conference. Brisbane, Queensland. 3 - 5 October 2002.

- I.A.P.D. Congress. New Orleans, U.S.A. 16 - 19 October 2003.

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